

SHORT CASES IN CARDIOLOGY

Abnormal atrial and ventricular repolarisation resembling myocardial injury after tricyclic antidepressant drug intoxication

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A 44 year old woman was admitted to our intensive care unit five hours after ingestion of amitriptyline. She was comatose (Glasgow coma score 3) with dilated pupils non-reactive to light and accommodation. Biochemical findings and serum electrolytes were normal. Arterial blood gas measurements revealed a slight metabolic acidosis. She was immediately intubated and mechanically ventilated. Gastric lavage was performed for gastrointestinal

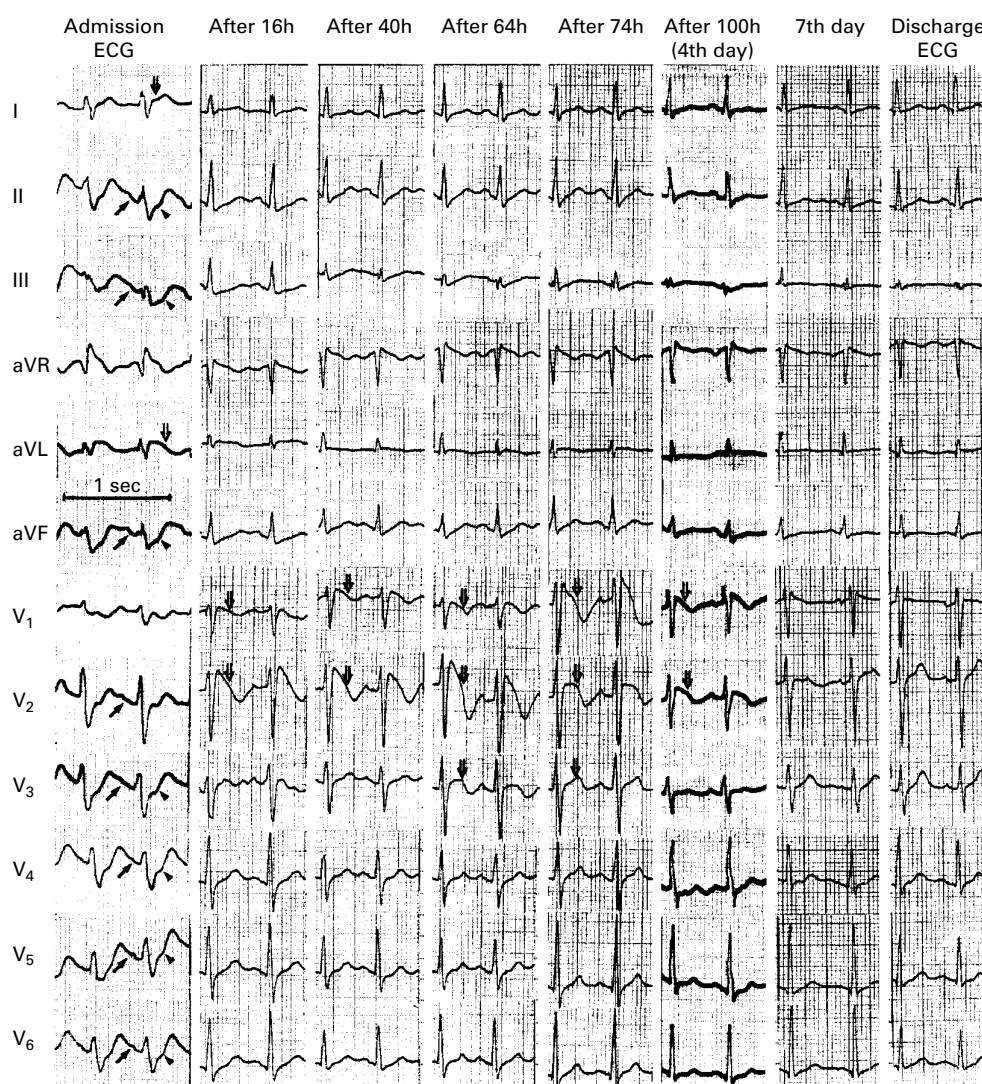


Figure 1 Serial ECGs recorded during hospitalisation. Admission ECG shows abnormal atrial and ventricular repolarisation. PR segment is depressed in II, III, aVF, V2 to V6 (small arrows), whereas ST segment elevation is evident in I and aVL (large arrows) with reciprocal ST depression in II, III, aVF, and V3 to V6 (arrowheads). The initial alterations do not exist in the second ECG, but new ST elevation with T wave inversion is obvious in V1 to V3 (large arrows). These become more prominent over time and persist for four days.

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decontamination followed by the administration of activated charcoal. She recovered after 10 days in the intensive care unit with no apparent neurological sequelae.

The admission ECG showed sinus tachycardia (125 beats/min) with a few supraventricular premature beats, QRS interval widening of 150 ms resembling right bundle branch block (RBBB), and a prolonged QT interval (QT 360, corrected QT (QT_c) 520 ms). ST segment elevation was noted in the left limb leads (I, aVL) (fig 1). After 16 hours, a new ST segment elevation was noted in precordial leads V1 and V2. Serial ECGs showed abnormal ventricular repolarisation characterised by ST elevation and T wave inversion in right precordial leads, mimicking a current of anteroseptal subepicardial injury, which persisted for four days. The QT_c returned to normal on the third day. QRS duration remained > 100 ms until the fourth day. The terminal 40 ms of the frontal plane QRS vector (T40ms) was > 120° for seven days, while incomplete RBBB was evident for eight days. Supraventricular ectopy lasted for only two hours from initial presentation, and ventricular arrhythmias were never recorded. ECG at discharge from the intensive care unit was normal (fig 1).

Serial cross sectional colour echo Doppler evaluation revealed no abnormalities. In particular global and segmental kinesis of both ventricles were normal and there was no evidence of pericardial effusion. Myocardial enzymes (creatinine kinase (CK), CK isoenzyme MB, troponin T) and electrolytes were normal.

At presentation, serum amitriptyline (4880 ng/ml) greatly exceeded the upper limit of the therapeutic range (300 ng/ml). After 16 hours serum amitriptyline was decreased to 1310 ng/ml and remained constant for some days, gradually falling towards therapeutic concentrations by day 10 (1324 day 2; 1108 day 5; 653 day 7; 322 day 10).

Our patient had clinical features of severe antidepressant drug intoxication. ECG abnormalities such as sinus tachycardia, notably widened QRS interval resembling RBBB, T40ms > 120°, and prolonged QT_c are usual in these patients.^{1,2} However, pronounced ECG alterations concerning the repolarisation of atria and ventricles resembling acute infarction are not only unusual^{3,4} but to our knowledge have never been simultaneously registered in a patient. In addition, migrating ST elevation to another territory with abnormally inflected T wave also mimicking subepicardial injury has never been reported before. The excessively increased amitriptyline serum concentrations contributed to the appearance of these unusual ECG abnormalities.

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